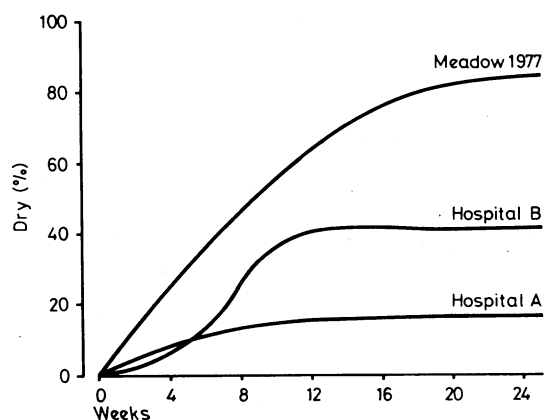


the buzzer alarm, and the figure for hospital B was 33 %. By far the commonest difficulty was failure to awake to the alarm. Most of these patients stopped using the buzzer altogether because of the difficulties, most of which occurred in the first week of using the buzzer alarm.

The percentage of patients who could be said to have been cured of their nocturnal enuresis was only 17 % at hospital A and 41 % at hospital B. The figure compares the effectiveness of the buzzer in this study with Meadows's results.¹



Rate at which children became dry using enuresis alarm. Comparison of results at hospitals A and B with Meadows's results.¹

Comment

Many papers have shown how the buzzer alarm should be used.¹⁻⁴ With this knowledge it is possible to determine the reasons for failure of the buzzer to cure enuresis in my study: (1) none of the paediatricians took a special interest in enuretics; (2) the use of the buzzer was not satisfactorily demonstrated before issue; (3) follow-up appointments were not frequent enough; and (4) many patients were seen by a different doctor each time they attended the clinic.

Nocturnal enuresis is a common problem, and effective management should be provided when the family seeks medical advice. Failure to do so may cause the family to lose faith in their doctors and lead to many years of misery for the child. I think that the poor results of my study may be repeated at many hospitals in the United Kingdom. There is no point in children with enuresis attending hospital at three-monthly intervals, year after year, without any improvement. This was the case with an appreciable number of patients in this study. If general practitioners have such patients then they should consider purchasing their own buzzer alarms for the practice. The general practitioner is in an ideal position to manage such children. He has often known the family for years and the child since birth. From this knowledge he is able to judge whether the child is mature enough to cope with a buzzer. He will know the social circumstances of the family and the sleeping arrangements of the children. All these factors are important if the buzzer is to be used successfully. If the practice has an interested health visitor she would be invaluable in helping with home visits.

The extra work would be compensated for by the satisfaction of successfully managing what can be a difficult problem, and surely this role for the general practitioner would be in keeping with the spirit of the Court Report⁵—which coined the term general practitioner paediatrician.

¹ Meadow SR. How to use buzzer alarms to cure bed-wetting. *Br Med J* 1977;ii:1073-5.

² Dische S. In: Kolvin, MacKeith, Meadow, eds. *Bladder control and enuresis*. London: Spastics International Medical Publications, 1973.

³ Forsythe WI, Redmond A. Enuresis and the electric alarm: study of 200 cases. *Br Med J* 1970;i:211-3.

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⁵ Committee on Child Health Services. *Fit for the future*. London: HMSO, 1976.

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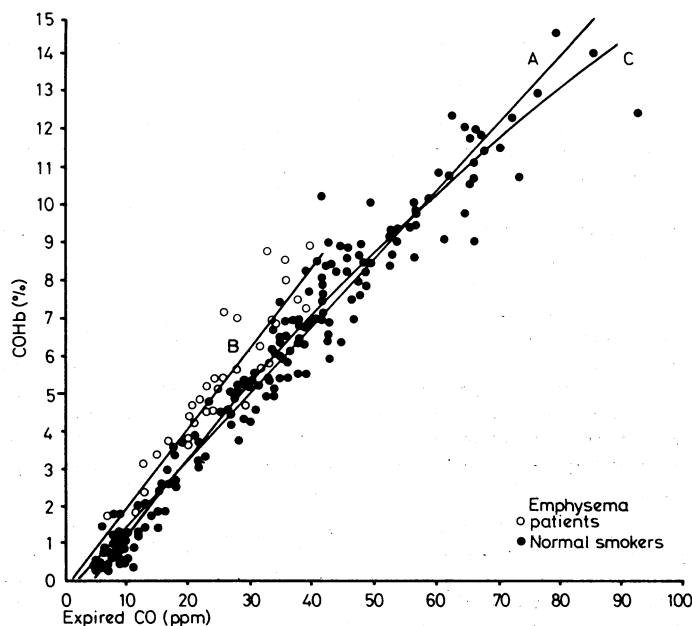
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Expired air carbon monoxide: a simple breath test of tobacco smoke intake

The amount of carbon monoxide in end-expired alveolar air provides a rapid and accurate non-invasive indirect measure of carboxyhaemoglobin,¹ since carbon monoxide in alveolar air after breath-holding is in equilibrium with the concentration of carboxyhaemoglobin in the blood. The carbon monoxide analyser (Ecolyzer Series 2000, Analysis Automation Ltd) is portable and inexpensive (£930). The method has a potentially wide application as an objective test of the amount smokers inhale and of their claims to have stopped. Although it has been available for several years, more cumbersome and costly methods that are no more accurate, and in some cases require blood samples, continue to be advocated. This paper shows the accuracy of the method and that error due to impairment of lung function among smokers is negligible for most purposes.

Subjects, methods, and results

Parallel venous blood and expired air samples were obtained from two groups of subjects: (1) "normal smokers" from the smokers' clinic at the Addiction Research Unit who gave no history of chronic lung disease, though this was not specifically excluded; (2) patients with emphysema from King's College Hospital chest unit who had been diagnosed on radiological criteria.² Expired alveolar air samples were collected by the method of Jones *et al.*³



Scattergram of expired air carbon monoxide (CO) against carboxyhaemoglobin (COHb). Linear regression lines: (A) for normal smokers; (B) for emphysema patients. (C) indicates line derived from the multiple regression analysis for smokers of average age for sample (39.6 years). Corresponding regression equations: (A) $\text{COHb} = -0.28 + 0.175 \text{ ECO}$; (B) $\text{COHb} = -0.12 + 0.211 \text{ ECO}$; (C) $\text{COHb} = -1.629 + 0.222 \text{ ECO} - 0.0006 \text{ ECO}^2 + 0.018 \text{ age}$. Note ECO refers to linear and ECO^2 to quadratic expired air components.

The subjects were instructed to exhale completely, then to take a deep breath, hold it for 20 seconds, and breathe out rapidly through a three-way connector into, firstly, a 500-ml anaesthetic bag (to allow for the dead space) and, secondly, an impermeable plastic bag⁴ to provide the sample for immediate measurement by the Ecolyzer. The time between sample collection and the last cigarette was never under four minutes. The instrument was calibrated daily with a mixture of 80 parts per million carbon monoxide in air. The blood samples were analysed by an IL 282 CO-Oximeter. The accuracy of this method was checked by comparison with a gas chromatographic technique,⁵ yielding a correlation of 0.99 for 43 specimens over the range 0.6%-12.3% carboxyhaemoglobin. Repeated measurements by CO-Oximeter at four concentrations of carboxyhaemoglobin saturation gave the following values (mean \pm SE, $n=5$): $0.52 \pm 0.06\%$, $4.12 \pm 0.02\%$, $8.12 \pm 0.02\%$, and $14.72 \pm 0.06\%$.

The correlation between the expired air carbon monoxide reading (ECO)

and carboxyhaemoglobin was 0.98 ($n = 182$) for the normal smokers (standard error of estimate of carboxyhaemoglobin from a given ECO was 0.76%) and 0.92 ($n = 35$) for the patients with emphysema (figure). The slopes of the two regression lines were significantly different ($F = 5.8$; $df\ 33, 180$; $p < 0.001$). A stepwise multiple regression analysis of the data from the normal smokers showed, in addition to the linear ECO component, a significant quadratic ECO component and a significant age component.

Comment

The difference between the slopes of the linear regressions of the normal smokers and the patients with emphysema shows that impaired lung function affects the relationship between expired air carbon monoxide and carboxyhaemoglobin. In emphysema a given concentration of expired air carbon monoxide is associated with a higher carboxyhaemoglobin, reflecting impaired diffusion of carbon monoxide from blood into the alveoli. The small age effect found in the multiple regression should also be interpreted in this way. Nevertheless, impaired lung function cannot be an important factor in normal smokers, since only 3% of the variation in carboxyhaemoglobin remained unaccounted for after allowing for linear and quadratic ECO components and age. The closeness of the relationship between carboxyhaemoglobin and expired air carbon monoxide suggests that there is little to be gained from analysing a sample of blood rather than expired air. Indeed, there is likely to be as much variation between different blood measures for carboxyhaemoglobin as was found for the two measures in the present study.

The expired air method has numerous advantages. It is non-invasive, portable, cheap, quick, and requires no specialist technical back-up. Because it is quick the results can immediately be fed back and explained to the smoker, which may well have an appreciable motivating effect. Smokers' claims to have stopped smoking can be instantly checked. It also has potential as a measure of smoke intake in epidemiological studies.

We thank Dr D Hutchinson for allowing us to study patients under his care and for his helpful comments. Financial support was provided by the Medical Research Council.

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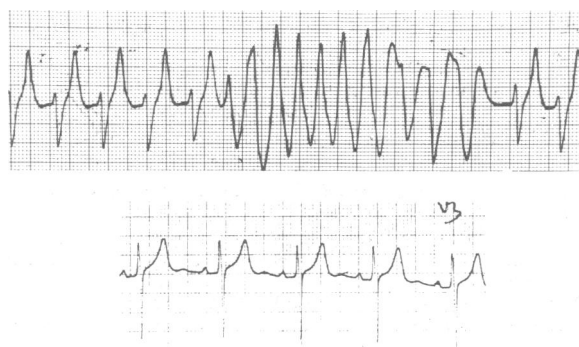
Rapid poisoning with slow-release potassium

Tablets containing slow-release potassium are widely prescribed but seldom taken in overdose. We report three cases in which life-threatening hyperkalaemia developed rapidly.

Case reports

Case 1—A 36-year-old man took an unknown number of tablets of hydralazine and Navidrex K (cyclopentiazide 0.25 mg with slow-release potassium chloride 600 mg) after drinking heavily. About five hours later he was vomiting frequently, his pulse was 125/min and blood pressure 130/90 mm Hg.

The electrocardiogram showed sinus rhythm with wide QRS complexes, tall T waves, and short runs of ventricular tachycardia (figure). The plasma potassium concentration was 8.9 mmol (mEq)/l and urea concentration was 6 mmol/l (36 mg/100 ml). He was given intravenous insulin, dextrose, calcium gluconate, and sodium bicarbonate, and oral calcium polystyrene sulphonate, with intravenous fluids and frusemide to force a diuresis. The plasma potassium concentration fell to 7.6 mmol/l after 30 minutes and to 6.1 mmol/l after a further 50 minutes but rose one hour later to 6.4 mmol/l. More dextrose, insulin, and frusemide were given and after five hours' treatment the plasma potassium concentration was 4.4 mmol/l. By this time he felt well and the electrocardiogram soon returned to normal. During the diuresis the urine output was about 1 l/h with potassium concentrations of 24 mmol/l to 43 mmol/l.



Electrocardiogram after overdosage of potassium chloride and cyclopentiazide (case 1). Upper strip from monitor 5 hours after overdosage (plasma potassium concentration 8.9 mmol/l); lower strip lead V3 16 hours after overdosage (plasma potassium concentration 5.0 mmol/l).

Case 2—A 58-year-old woman took about 20 tablets of Neo-NaClex-K (bendroflumazide 2.5 mg with slow-release potassium chloride 630 mg) and some phenylbutazone. Vomiting started after 30 minutes and she became sweaty and breathless. On admission five hours after ingestion she was in left ventricular failure, with cyanosis, widespread lung crepitations, a regular pulse of 72/min and blood pressure 110/80 mm Hg. The plasma potassium concentration was 8.0 mmol/l and urea concentration 3.7 mmol/l (22 mg/100 ml). She was given oxygen and intravenous frusemide, dextrose, and insulin. Gastric lavage recovered some tablet fragments and calcium polystyrene sulphonate was left in the stomach. By six hours after ingestion the plasma potassium concentration had risen to 9.1 mmol/l but further frusemide, insulin, and dextrose reduced it to 7.9 mmol/l at seven hours, 6.2 mmol/l at nine hours, and 3.9 mmol/l at 24 hours. The electrocardiogram showed left bundle-branch block which persisted after recovery, but the initial tall T waves became much smaller. The cardiac failure cleared rapidly but she remained nauseated for three days. Liver function tests and cardiac enzymes were normal.

Case 3—A 26-year-old man took 10 tablets of Distalgesic (dextropropoxyphene and paracetamol) and about 40 Slow-K tablets (slow-release potassium chloride 600 mg) and soon started vomiting. On admission three and a half hours after ingestion he was conscious with a regular pulse of 75/min and blood pressure 120/90 mm Hg. During gastric lavage he had a cardiac arrest with asystole. Resuscitation (including cardiac pacing) was unsuccessful. Plasma potassium concentration was 9.3 mmol/l and urea concentration 3.6 mmol/l (21 mg/100 ml) but dextropropoxyphene was not detected.

Comment

Severe hyperkalaemia due to oral potassium is said to be rare unless renal function is appreciably impaired.^{1,2} Hyperkalaemia occurred rapidly in our patients, however, even in two who also took a thiazide diuretic. The plasma potassium concentration and electrocardiogram must be monitored in every case of potassium overdosage. Electrocardiographic signs of hyperkalaemia (tall peaked T waves, PR prolongation, disappearance of P waves, QRS widening, heart block) are indications for immediate treatment. Intravenous dextrose (50 g) and soluble insulin (15 units) should be given, with sodium bicarbonate to correct any metabolic acidosis and reduce the extracellular potassium concentration. Calcium gluconate (10-20 ml of a 10% solution intravenously) reduces the risk of cardiac dysrhythmias and cardiac pacing may be helpful if severe bradycardia occurs.³ Intravenous fluids and frusemide should be given to force a diuresis and increase potassium excretion, but peritoneal or haemodialysis may be needed if renal function is impaired.